Available online at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevier.com/locate/ihj

Case Report

Recurrent A V block following ablation for AVNRT

Fnu Shailesh[*], *Asif Sewani*, *Hakan Paydak*

Department of Cardiovascular Medicine, University of Arkansas for Medical Sciences, 4301, W Markham St, # 634, Little Rock, AR 72205-7199, USA

ARTICLE INFO

Article history:
Received 8 February 2014
Accepted 9 October 2014
Available online 30 October 2014

Keywords:
Atrio-ventricular nodal re-entrant tachycardia (AVNRT)
Slow pathway (SP)
Heart block
Radiofrequency ablation (RFA)
Electrocardiogram (ECG)

ABSTRACT

Slow pathway modification has become the mainstay for the treatment of atrio-ventricular nodal re-entrant tachycardia (AVNRT) ablation because of high success rate and low incidence of complications. Our patient had a rare complication of slow pathway modification by radiofrequency ablation (RFA) in form of delayed complete heart block, occurring 10 days after the procedure and resolving in 6 weeks to normal conduction. Complete AV block is a rare immediate complication of RFA but can present weeks later. Transient atrio-ventricular (AV) block during the procedure is seen in all patients who develop delayed AV block and these patients should be monitored closely.

1. Introduction

Slow pathway modification is the preferred treatment for AVNRT due to high success and low complications. Reversible complete AV block is seen during slow pathway modification. Permanent AV block is due to permanent injury and is seen in immediate post-operative period. We present a case showing recurrent AV block after slow pathway modification, which improved after 4–6 weeks.

2. Case report

A 55 year old African–American male presented to the emergency room with complaints of palpitations and dizziness for 3 days. He was having these episodes for 2 years and was diagnosed with paroxysmal supraventricular tachycardia (PSVT). Patient initially wanted to be treated with medications and was taking beta-blocker (metoprolol) but he was having these episodes more frequently. In emergency room, his vitals were normal but his heart rate increased to 150 beats/min during one of his episodes with no change in blood pressure. Rest of his physical exam was normal.

Electrocardiogram (ECG) during the episode of palpitations showed narrow complex tachycardia with short RP interval. His ECG at baseline did not show any pre-excitation. His blood test including complete blood count, electrolytes, kidney function test and serum thyroid stimulating hormone (TSH) levels were normal. Patient was admitted to the hospital and overnight telemetry showed atrial couplets and triplets initiating these episodes. During the supraventricular tachycardia, patient had ventricular couplets which did not reset the tachycardia cycle length. Patient had treadmill stress

* *Corresponding author*. Tel.: +1 5016506624; fax: +1 5016865609.
E-mail addresses: drshailesh1982@gmail.com, fshailesh@uams.edu (F. Shailesh).
http://dx.doi.org/10.1016/j.ihj.2014.10.410

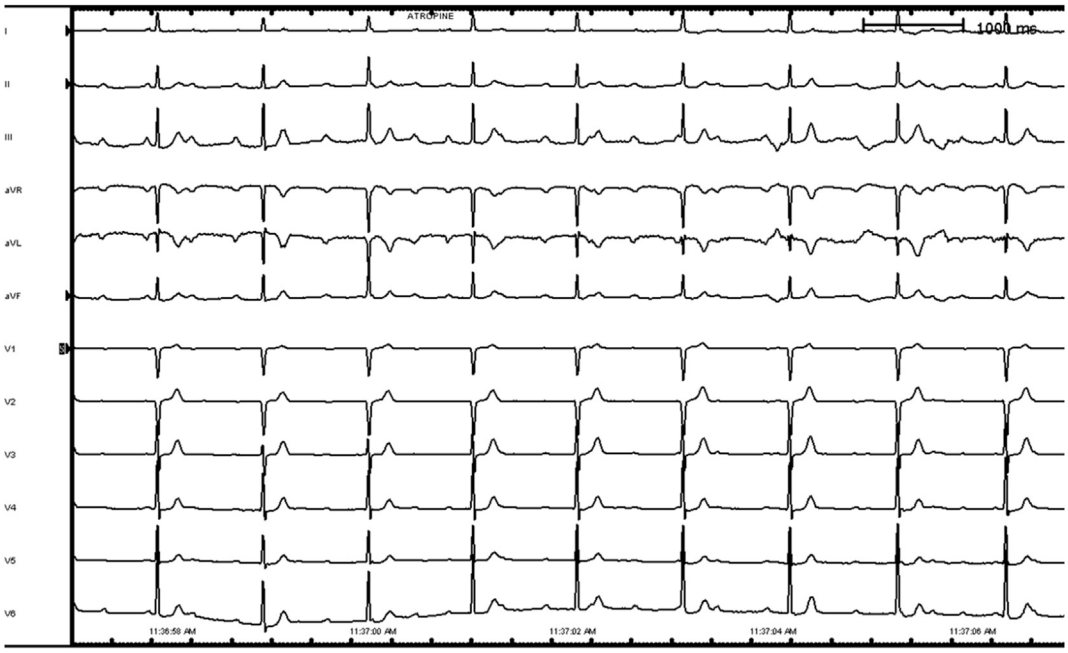

Fig. 1 – ECG showing complete AV block during radio-frequency ablation.

echocardiogram and transthoracic echocardiogram done previous year, which showed normal left ventricular size and function without stress-induced ischemia.

Patient was willing for an ablation procedure during this admission and electrophysiological study was scheduled for next day. During the procedure patient was in sinus rhythm with AA interval of 640 ms, AV interval 641 ms, QRS duration 65 ms, PR interval 180 ms, AH interval 75 ms and HV interval 41 ms. Right ventricular apical pacing showed decremental retrograde conduction without any right or left accessory pathway. Right ventricular apical pacing at 350 ms showed typical AVNRT (down the slow and up the fast). Radio-frequency ablation (RFA) was started for slow pathway modification. While ablating the slow pathway with 9th RF application, patient developed complete heart block [Fig. 1] which later became 2:1 atrio-ventricular (AV) block. RFA was stopped in less than 3 s and patient was treated with gluco-corticoids, isoproterenol and atropine. His rhythm changed to Mobitz type 1 block and in 1 h he was conducting 1:1 with prolonged PR interval [Fig. 2]. Post ablation, high right atrial

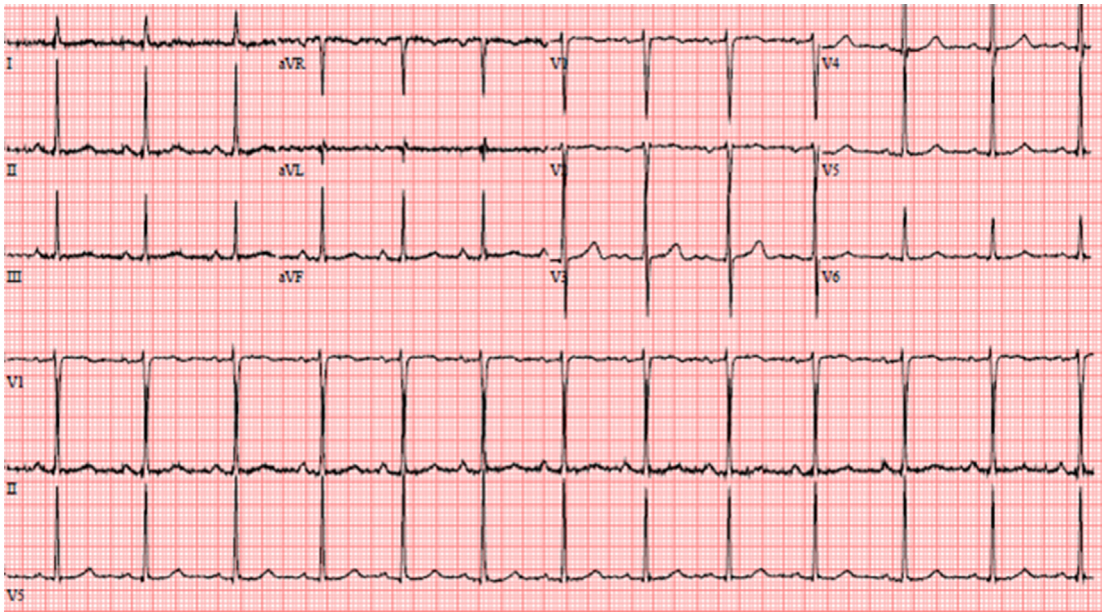

Fig. 2 – ECG showing normal sinus rhythm with 1:1 AV conduction after AVNRT ablation.

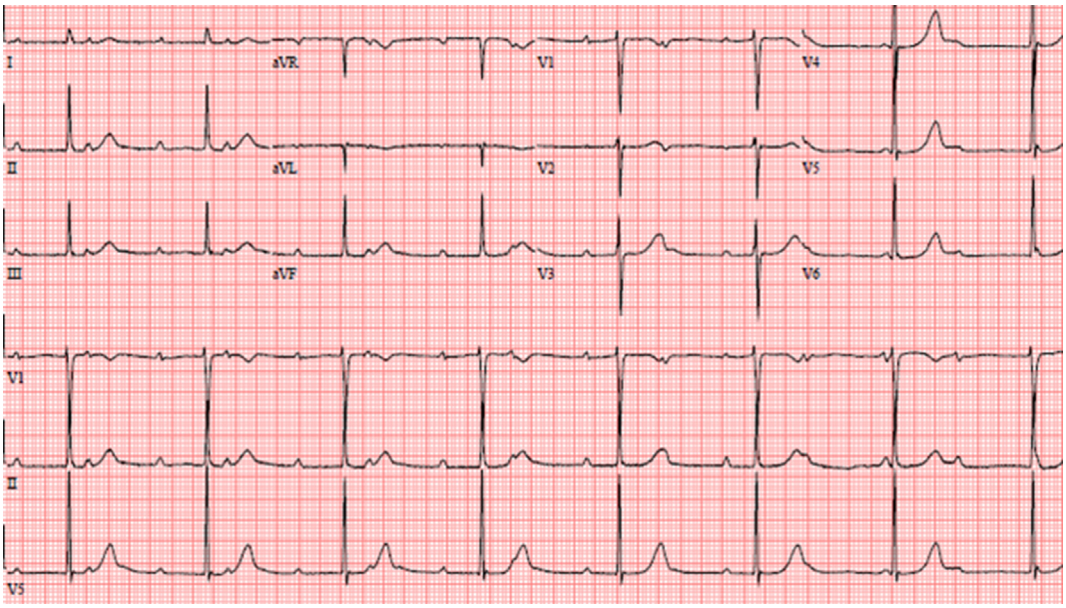

Fig. 3 – ECG showing complete AV block, 10 days after AVNRT ablation.

pacing revealed normal AV conduction with AH wenckebach periodicity of 300 ms. Patient was observed overnight and he continued to have normal AV conduction. He was discharged home next day with routine follow up.

Patient presented to emergency room 10 days later with complaints of shortness of breath, fatigue and dizziness. His vitals and physical exam was normal. His ECG showed sinus rhythm with complete heart block and junctional escape [Fig. 3], which did not respond to atropine. His blood tests, including electrolytes were normal. His AV block did not improve for next 24 h and was considered secondary to AV nodal scarring due to complication of RF ablation. Dual chamber pacemaker was installed in view of unresolving AV block. Patient was discharged and followed up in clinic. During his clinic visit in 6 weeks, patient was found to have sinus tachycardia with 1:1 AV conduction [Fig. 4].

Our patient had a rare complication of slow pathway modification by RF ablation in form of delayed complete heart block which resolved in 6 weeks to normal conduction.

3. Discussion

AVNRT is the most common paroxysmal supraventricular tachycardia which originates from reentry circuit in slow and fast pathway of AV node. AV conduction has been shown to be

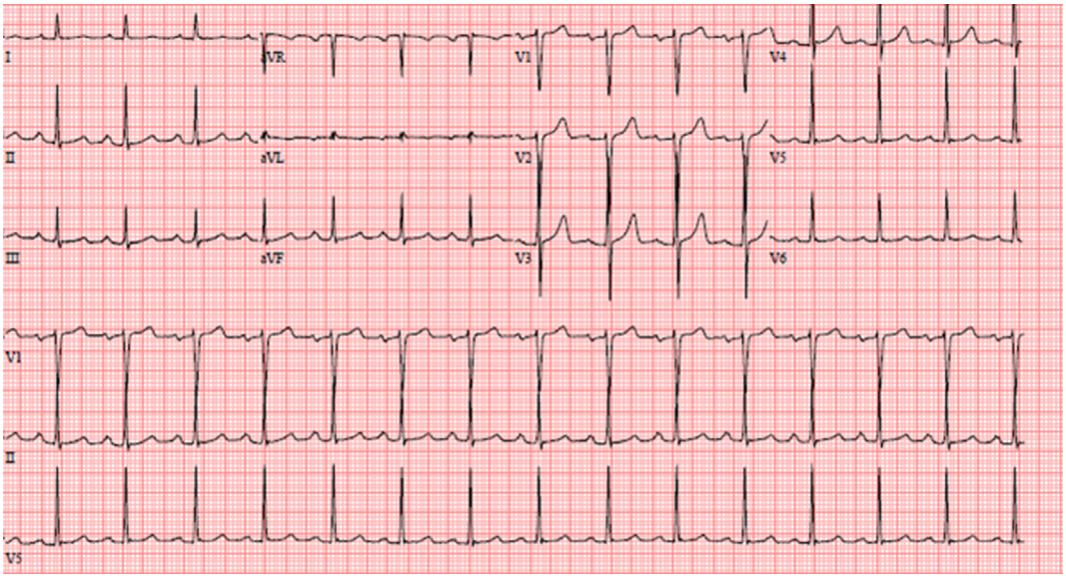

Fig. 4 – ECG at 6 week follow up, showing complete recovery of AV conduction.

sufficiently maintained by fast pathway after blocking slow pathway by RFA or cryotherapy.[1] Slow pathway modification has been a very effective treatment for AVNRT, with success rates >99%.[2]

Complete AV block is a rare complication of AVNRT. It is more commonly associated with fast pathway ablation (5–6%) as compared to slow pathway modification (1–2%).[3] Persistent high degree AV block is thought to be due to permanent injury due to RFA/cryotherapy. Most of the cases are transient and occur during the procedure. AV conduction is affected as result of edema caused by RFA/cryotherapy in tissue around AV node. AV conduction recovers after edema resolves.

Delayed AV block has only been reported in few case reports. Fenelon et al reported a patient with delayed impairment in AV conduction (Mobitz type 1 block at rest and Mobitz type II block during exercise), 3 months after slow pathway modification.[4] Williamson et al reported 3 cases of complete AV block, which developed 36–72 h after the slow pathway ablation procedure.[5] All of these cases had transient AV block during the procedure, which might be useful as a predictor of delayed AV block. However, duration of AV block during procedure did not predict development of delayed AV block. Brembilla et al reported 3 patients with completed AV block, which developed 1–3 days after slow pathway modification and resolved in 7 days. These patients had 1:1 conduction with first degree AV block at 1 year.[6]

Cause of delayed AV effects of RFA has not been clear but few mechanisms have been proposed. RFA produces coagulative necrosis surrounded by inflammation, which usually resolves. In some cases inflammation might lead to fibrosis and lesion extension secondary to micro-circulatory damage.[7]

4. Conclusion

Complete AV block is an immediate complication of RFA but can rarely present days to weeks after RFA. Patients having transient AV block during the procedure should be monitored closely. Implantation of permanent pacemaker is safer in these patients, since there is no way of predicting patients with favorable outcome.

Conflicts of interest

All authors have none to declare.

REFERENCES

1. Csanadi Z, Klein GJ, Yee R, Thakur RK, Li H. Effect of dual atrioventricular node pathways on atrioventricular reentrant tachycardia. *Circulation*. 1995;91:2614–2618.
2. Kay GN, Epstein AE, Dailey SM, Plumb VJ. Role of radiofrequency ablation in the management of supraventricular arrhythmias: experience in 760 consecutive patients. *J Cardiovasc Electrophysiol*. 1993;4:371–389.
3. Hindricks G. Incidence of complete atrioventricular block following attempted radiofrequency catheter modification of the atrioventricular node in 880 patients. Results of the Multicenter European Radiofrequency Survey (MERFS) the Working Group on Arrhythmias of the European Society of Cardiology. *Eur Heart J*. 1996;17:82–88.
4. Fenelon G, d'Avila A, Malacky T, Brugada P. Prognostic significance of transient complete atrioventricular block during radiofrequency ablation of atrioventricular node reentrant tachycardia. *Am J Cardiol*. 1995;75:698–702.
5. Williamson BD, Man KC, Daoud E, Niebauer M, Strickberger SA, Morady F. Radiofrequency catheter modification of atrioventricular conduction to control the ventricular rate during atrial fibrillation. *N Engl J Med*. 1994;331:910–917.
6. Brembilla-Perrot B, Beurrier D, Jacquemin L, et al. Complete atrioventricular block, a possible complication of radiofrequency ablation of reciprocating nodal tachycardia. *Arch Mal Coeur Vaiss*. 1996;89:729–734.
7. Fenelon G, Brugada P. Delayed effects of radiofrequency energy: mechanisms and clinical implications. *Pacing Clin Electrophysiol*. 1996;19:484–489.